RESPIRATORY INTENSIVE CARE

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Little, Brown and Company
Boston/Toronto

To my wife Ann
and children Francis, Kenneth, Katy,
Melissa, Mark, Glen, Timothy,
Sheila, Meghan, and Bridget

To my wife Penny
and children Kaitlin and Patrick

To my wife Sylvia
and son Peter

First Edition

Library of Congress Catalog Card No. 86-80720

ISBN 0-316-54193-1

Printed in the United States of America

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FOREWORD

Critical care medicine is becoming increasingly complex. This book *Respiratory Intensive Care* provides information that should make the task of providing good care for the respiratory patient easier. This book is unique because it focuses predominantly on the respiratory patient. Only patients with a primary respiratory disorder such as chronic obstructive lung disease, asthma, pneumonia, and the adult respiratory distress syndrome are discussed. This allows the authors to be less global and more specific in their approach. Complications such as arrhythmias are reviewed but are discussed as they affect the patient who has a primary disease of the respiratory system. The editors have skillfully constructed the book in such a way that respiratory pathophysiology is first covered. An approach to the patient at the bedside with a respiratory disorder is then described in relationship to the previously described discussion of respiratory pathophysiology. Specific recommendations are made that allow appropriate clinical decision making. For example, in the nutrition section, recommendations are tailored for the respiratory patient. Additionally, specific recommendations are made for mechanical ventilation for specific groups of respiratory patients.

This book should be useful to clinicians who provide care to respiratory patients who are critically ill. Important information is available for physicians, respiratory therapists, and nurses as well as the medical student because the book is organized in such a way to provide a basic framework for the understanding of respiratory disease and the critically ill respiratory patient.

Roger C. Bone

PREFACE

An increasing number of patients require intensive care each year in the United States. Many of these patients have a primary disorder of the respiratory system necessitating intensive therapeutic intervention, while an additional large number have nonpulmonary diseases complicated by respiratory failure. Clinicians are, therefore, being confronted by an increasing number of patients experiencing acute respiratory decompensation. For example, chronic obstructive pulmonary disease now is the sixth leading cause of death in the United States, while asthma afflicts approximately 2.5 percent of the population.

The goal of this book is to provide clinicians (physicians, nurses, and respiratory therapists) at all levels of training with a comprehensive, though not encyclopedic, structured framework for assessing and instituting care for patients with acute respiratory problems in the intensive care unit. The general format is designed to present those basic physiologic principles that are needed in order to formulate a rational therapeutic plan. Principles of mechanical ventilatory support are then described in detail along with a practical guide for its institution. A discussion of specific respiratory diseases follows with an emphasis on pathophysiologic principles and practical therapeutic approaches. Issues of special interest in the treatment of respiratory failure such as hemoptysis, aspiration, pneumothorax, and clotting disorders are discussed in separate chapters. Because of the increasing complexity of nursing responsibilities and the many legal questions now faced by clinicians caring for patients in the intensive care unit, these issues are specifically discussed.

The book features an appendix with a discussion of arterial blood gases followed by a detailed schema of the most popular mechanical ventilators.

The methods and approaches to intensive respiratory care represent those used by present and past clinicians and former trainees of the Tufts Lung Station at St. Elizabeth's Hospital of Boston.

We wish to thank our colleagues Charles Hales, M.D., Richard Matthay, M.D., Gustave Laurenzi, M.D., Joseph Andrews, M.D., and Theodore Smith, M.D. for their review of portions of this text and their constructive comments.

A special note of appreciation goes to Judy O'Rourke and Lynn Sullivan for their diligent, tireless efforts in the preparation of this manuscript.

Jim Krosschell, Lou Bruno, and the staff at Little, Brown and Company were of invaluable assistance.

The support of the St. Elizabeth's Hospital Pulmonary Research Fund (Harold Alfond/Dexter Shoes, Beatrice Flynn, Robert Martin, Joan Timmons, William Madden, Beatrice Gerety, James Milligan, and Jeanne Skelton) is greatly appreciated.

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CONTENTS

I. PHYSIOLOGIC PRINCIPLES AND TECHNIQUES OF RESPIRATORY INTENSIVE CARE

Notice

The indications and dosages of all drugs in this book have been recommended in the medical literature and conform to the practices of the general medical community. The medications described do not necessarily have specific approval by the Food and Drug Administration for use in the diseases and dosages for which they are recommended. The package insert for each drug should be consulted for use and dosage as approved by the FDA. Because standards for usage change, it is advisable to keep abreast of revised recommendations, particularly those concerning new drugs.

Patrick J. Fahey

1. PRINCIPLES OF OXYGENATION IN THE CRITICALLY ILL

Patients requiring intensive care secondary to respiratory disorders present the spectrum of causes of arterial hypoxemia. Hypoventilation (from drug overdose or myasthenia gravis), ventilation perfusion disturbance (caused by asthma or chronic obstructive pulmonary disease [COPD]), and intrapulmonary shunting (from cardiac and noncardiac pulmonary edema) can cause inadequate tissue oxygenation through desaturation of hemoglobin. Decreases in cardiac output or increases in oxygen consumption occur regularly in the critically ill patient and threaten further the adequacy of cellular oxygenation. Appropriate therapy for the respiratory patient requires knowledge of oxygen delivery and utilization, identification of the physiologic cause of hypoxemia through calculation of the alveolar-to-arterial oxygen gradient, and noting the response of hypoxemia to oxygen therapy. This chapter reviews the principles of tissue oxygenation, oxygen delivery, and consumption and provides a physiologic rationale for therapeutic approaches to tissue and alveolar hypoxia.

CELLULAR OXYGENATION

Each cell in the body is responsible for meeting its own energy needs. Normal cellular function is maintained through each cell's ability to supply the required amount of energy for performance of its special function. The basic energy-producing process within all cells is oxidative phosphorylation, a process in which electrons derived from the Krebs cycle pass in "bucket brigade" fashion down a series (15–20 steps) of oxidative reductive reactions. The final step of this process involves transfer of electrons to molecular oxygen by the activating enzyme cytochrome oxidase A_3. The transit of electrons from start to finish through the bucket brigade produces more than 50 kcal of energy per mole of water formed. Most of this energy is trapped and stored in a freely diffusible form, adenosine triphosphate (ATP), available for use throughout the cell.

The flow of electrons results in steady production of ATP, which is vital for normal cellular and tissue function. This flow requires that a continuous supply of oxygen be made available within the mitochondria of each cell. Estimates of the critical amount of oxygen required for normal production of ATP indicates a value near 1 to 3 mm Hg within the mitochondria [1]. Levels below this figure disrupt the normal aerobic process, resulting in a conversion to anaerobic production of energy, a less efficient and limited process. Disruption or limitation in the normal supply of oxygen to tissues, which occurs frequently in critically ill patients, poses a constant threat to normal cellular metabolism. Thus a primary goal in the care of most critically ill patients is the maintenance of adequate levels of oxygen within body cells.

Body cells of a healthy individual at rest require approximately 250 ml of oxygen per minute for normal cellular aerobic function. This value represents the oxygen demand of all aerobic cellular constituents. In health, the oxygen demand of cells equals the oxygen consumption ($\dot{V}O_2$). Significant decreases in $\dot{V}O_2$, below 250 ml/minute (i.e., oxygen demand exceeds $\dot{V}O_2$) result in decreased aerobic energy production, a decline in cellular function, and the onset of anaerobic metabolism. Ultimately tissue dysfunction occurs if anaerobic metabolism is prolonged and severe enough. Tissue hypoxia, acidosis, and cellular death are the end results. An adequate cellular oxygen level is primarily a function of the balance of oxygen supply relative to oxygen demand. Because there are limits in the

ability to decrease the oxygen demand of tissues, primary therapeutic manipulations in patients threatened with inadequate tissue oxygen levels involve attempts to increase oxygen delivery.

DETERMINANTS OF OXYGEN DELIVERY

The amount of oxygen delivered to body tissues for use in aerobic metabolism is determined by three independent variables: the amount of hemoglobin, the percent saturation of hemoglobin with oxygen, and cardiac output. These variables represent the primary function of three separate organ systems: hematologic, pulmonary, and cardiovascular. Because these three systems interact to form the basis of aerobic life, they have been termed the grand architecture of physiology.

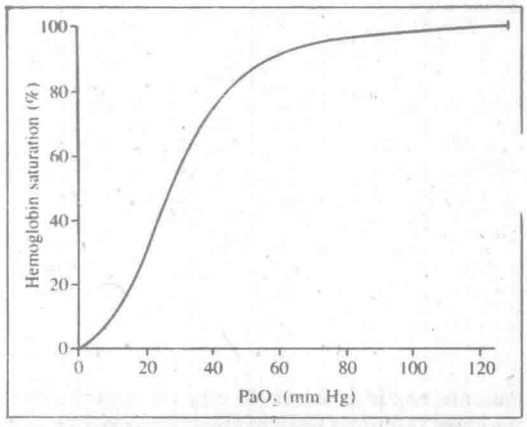

Fig. 1-1. Oxyhemoglobin dissociation curve for pH 7.40, temperature 37°C, and $PaCO_2$ 40 mm Hg.

1. *Hemoglobin.* Oxygen is transported in two forms in the blood: dissolved and combined with hemoglobin. Dissolved oxygen constitutes a tiny fraction of the total amount of oxygen in blood and can be determined by multiplying the partial pressure of oxygen in blood (PaO_2) by 0.003, the solubility coefficient for oxygen in plasma. A normal PaO_2 of 100 mm Hg indicates that only 0.3 ml of oxygen is dissolved per 100 ml of plasma (100 mm Hg × 0.003 ml/dl). If dissolved oxygen were the sole mechanism by which oxygen reached tissues, the cardiac output would have to be in the order of 80 liters/minute just to deliver enough oxygen to meet resting oxygen demands (0.3 ml/100 ml × 80,000 ml/minute = 240 ml/minute O_2 delivery.)

The second form of oxygen in blood—oxygen combined with hemoglobin—is by far the most important in terms of oxygen delivery. Hemoglobin, a marvelously complex yet functionally simple molecule, has evolved as a highly efficient transporter of oxygen. One gram of hemoglobin is capable of combining with 1.34 ml of oxygen. A normal hemoglobin value of 15 g/dl of blood indicates that more than 20 ml of oxygen is combined with hemoglobin in a 100-ml sample of blood when fully saturated. Threats of a reduction in oxygen content of arterial blood occur with a decreased hemoglobin level (as with anemia), abnormal hemoglobin structure, and carbon monoxide poisoning.

2. *Percent saturation of hemoglobin.* The remarkable oxygen-carrying capacity of hemoglobin relies on full saturation with oxygen. Saturation must occur as the red blood cells pass through the pulmonary capillary bed and abut the alveolar capillary membrane. Although PO_2 is responsible for only a tiny fraction of oxygen content, it is extremely important in determining the percent saturation of hemoglobin. Figure 1-1 describes the relation between the PO_2 and the percent saturation of hemoglobin. This well-known sigmoidal curve demonstrates that decreases in alveolar PO_2 result in decreases in hemoglobin saturation and, ultimately, decreases in O_2 content. It is notable in the curve that decreases in PaO_2 from more than 100 mm Hg to near 60 mm Hg result in only a modest 10 percent desaturation of hemoglobin. A 40 percent decrease in PaO_2 results in only a 10 percent fall in oxygen saturation. Nearly 90 percent of the normal oxygen content is maintained despite relatively large changes in the alveolar and plasma PO_2. Understanding the mechanisms of decreased alveolar O_2 is important for clinicians involved in pulmonary and critical medicine.

3. *Cardiac output.* The heart and blood vessels are responsible for delivery of the oxygen-laden hemoglobin to body tissues where oxygen diffuses from capillaries into cells. Venous blood is then returned to the heart and lungs and is pumped through the pulmonary capillary bed to reload with oxygen. Whereas compensations for decreased oxygen content in arterial blood can occur through increases in hemoglobin or cardiac output, there are poor compensatory mechanisms for declines in cardiac output. Although the heart and cardiovascular system play a vital role in the normal delivery of oxygen to tissues, they are at the same time the

weak link in the chain of oxygen delivery because of poor compensatory mechanisms.

DETERMINATION OF OXYGEN DELIVERY

Normal

The amount of oxygen delivered to body tissues via hemoglobin (Hgb) can be determined by the following relationship:

$$O_2 \text{ delivery} = \text{Hgb (g/dl)} \times 1.34 \text{ ml/g} \times \text{Hgb saturation}^* \times \text{cardiac output (ml/minute)}$$

Assuming normal values, then

$$O_2 \text{ delivery} = 15 \text{ g/dl} \times 1.34 \text{ ml/g} \times 0.97 \times 5000 \text{ ml/minute}$$

$$O_2 \text{ delivery} = 975 \text{ ml/minute}$$

Note that the equation determines oxygen delivery only for oxygen combined with hemoglobin; the dissolved portion is tiny and is ignored. Resting oxygen consumption is 250 ml/minute; thus at rest oxygen delivery to the tissues is greater than oxygen demand by a factor of nearly 4. This provides a reasonable reserve supply of vital oxygen for tissues.

Anemia

A decrease in hemoglobin to 7.5 g/dl, which might occur in a patient with gastrointestinal bleeding, has an effect on oxygen delivery. The above equation assumes that cardiac output and percent saturation remain unchanged.

$$O_2 \text{ delivery} = 7.5 \text{ g/dl} \times 1.34 \text{ ml/g} \times 0.97 \times 5000 \text{ ml/minute}$$

$$O_2 \text{ delivery} = 488 \text{ ml/minute}$$

A decrease in hemoglobin by 50 percent results in a decrease in oxygen delivery by 50 percent, and oxygen supply is only a factor of 2 greater than resting demands. Further decreases in oxygen delivery to values below one-half normal (approximately 450 ml/minute) may be associated with some cells being deprived of adequate oxygen, resulting in a conversion to anaerobic metabolism.

Clinical experience, however, indicates that significant anaerobic metabolism with accumulation of lactic acid does not occur with uncomplicated anemia even with extreme decreases in hemoglobin (e.g., Hgb 4 g/dl). This is because compensatory mechanisms, primarily increased cardiac output, attempt to maintain oxygen delivery near normal levels. Similarly, marked desaturation of hemoglobin, which occurs with alveolar hypoxemia caused by lung disease, is also seldom associated with lactic acidosis. In this instance, both increases in hemoglobin concentration and cardiac output compensate for decreases in arterial oxygen content and attempt to normalize oxygen delivery.

*Percent hemoglobin saturation should be expressed as a decimal fraction; for example, 97% = 0.97.

Cardiac Output

Tissue oxygen delivery is vitally dependent on cardiac output. Abrupt decreases in cardiac output, which might occur with acute myocardial infarction or arrhythmias, can lead to cellular hypoxia and anaerobic metabolism. Consider a 50-year-old person with normal hemoglobin and arterial PO_2 who suffers an acute myocardial infarction and subsequent decline in cardiac output.

$$O_2 \text{ delivery} = 15 \text{ g/dl} \times 1.34 \text{ ml/g} \times 0.97 \times 2500 \text{ ml/minute}$$

$$O_2 \text{ delivery} = 488 \text{ ml/minute}$$

The clinician faced with impending tissue hypoxia due to impaired myocardial function has limited therapeutic options. Attempts to normalize oxygen delivery by increasing hemoglobin levels with transfusions is contraindicated in the patient with a failing heart. The hemoglobin saturation is already 97 percent, leaving only minimal room for improvement; therefore therapy must be directed at improving myocardial performance.

OXYGEN DELIVERY AND OXYGEN DEMAND

Determination of oxygen delivery provides valuable information about the status of the respiratory, hematologic, and cardiovascular systems. Clinicians caring for critically ill patients, however, must regularly assess the adequacy of tissue oxygen delivery relative to oxygen demand. Although measurement of oxygen delivery (arterial oxygen content × cardiac output) supplies valuable information, it may lead to false assumptions if it is not related to oxygen demand. Consider the following example.

A 50-year-old man with a history of diabetes and

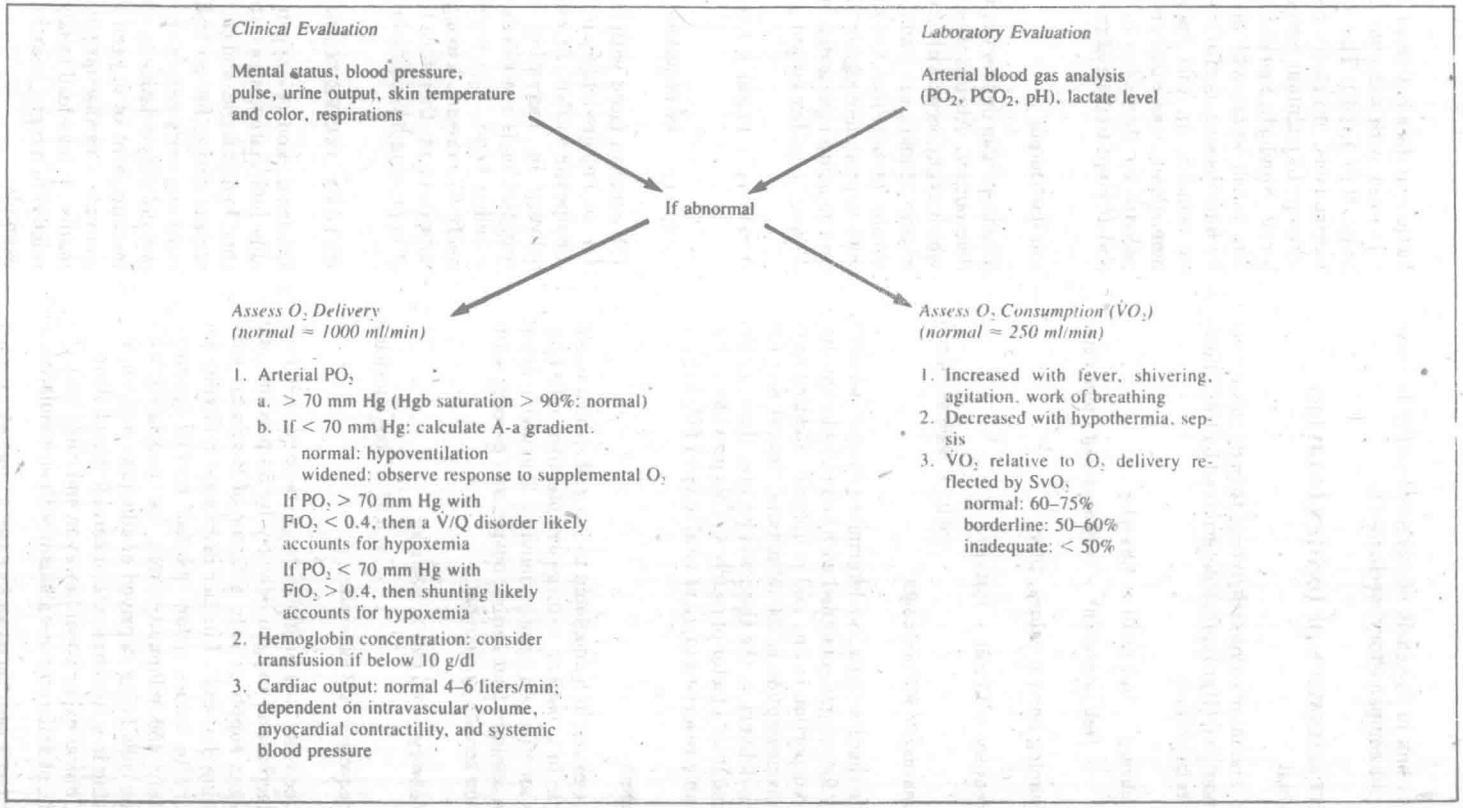

Fig. 1-2. Algorithm for assessing the adequacy of tissue oxygenation.